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Heart Disease Mortality Among Bridge and Tunnel Officers Exposed to Carbon Monoxide

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ABSTRACT

We investigated the effect of occupational exposure to carbon monoxide (CO) on mortality from heart disease in a retrospective study of 5529 New York City bridge and tunnel officers. Among former tunnel officers, 61 deaths from arteriosclerotic heart disease (ASHD) were observed, as compared to 45 expected [standardized mortality ratio (SMR) = 1.35, 90 percent confidence interval (CI) = 1.09, 1.68]; expected rates were based on the New York City population. Using a Cox proportional hazards model, we compared the risk of mortality from ASHD among tunnel officers to the less-exposed bridge officers. No association of ASHD with length of exposure was observed, but there was significant interaction of exposure with age. The elevated risk of ASHD among tunnel officers, as compared to bridge officers, declined after cessation of exposure with much of the risk dissipating within as little as five years. The parallel findings of this study of occupational exposure to CO and those studies showing the relationship of cigarette smoking to cardiovascular mortality suggest that CO may play an important role in the pathophysiology of cardiovascular mortality associated with cigarette smoking.

INTRODUCTION

Carbon monoxide (CO) may contribute to cardiovascular disease through several accepted and potential mechanisms[1,2] including binding to hemoglobin,[3,4] and reducing oxygen dissociation at the tissue level by shifting the oxygen-hemoglobin dissociation curve. Carbon monoxide in experimental animals also increases platelet stickiness[5] which may contribute to atherogenesis; and accelerates atherosclerosis either through altered lipid metabolism[6] or increasing vessel permeability to cholesterol.[6-8] Carbon monoxide in experimental studies contributes to sudden death by reducing the cardiac threshold for ventricular fibrillation.[9] Some of these mechanisms, such as those that would lead to transient hypoxia of the myocardium, would lead to increased risk of myocardial ischemia only when exposure is current, while other mechanisms e.g., acceleration of atherogenesis, may lead to long standing elevation of risk for cardiovascular disease.

The current Occupational Safety and Health Administration (OSHA) standard for CO for the protection of workers allows exposure of 50 parts per million (ppm) time-weighted-average (TWA) over an 8-hour day.[10] The margin of safety of this standard for workers with underlying cardiovascular disease is questioned by a series of studies of exposures averaging 50 ppm which have demonstrated a decrease in exercise necessary to induce angina in experimental subjects with underlying cardiovascular disease.[11-13] The National Institute for Occupational Safety and Health (NIOSH) recommends a standard for CO exposure

of 35 ppm as an 8-hour TWA with a ceiling limit of 200 ppm.[14] To assess the long-term effects of permissible levels of exposure to carbon monoxide on cardiovascular mortality in a working population, we studied a cohort of traffic control officers employed on bridges and in tunnels. In parallel to smoking cigarettes, which is associated with cardiovascular mortality[15-22] and which causes a rise in carboxyhemoglobin in the same order of magnitude as permissible occupational CO exposure,[2,23-25] we show that occupational exposure to CO also results in a significant excess of mortality from cardiovascular disease that remits on cessation of exposure.

METHODS

Population Identification

The study population consisted of all male bridge and tunnel officers (BTOs) employed between January 1, 1952 and February 10, 1981 at one of nine major water crossings (two tunnels and seven bridges) operated by the Triborough Bridge and Tunnel Authority (TBTA) of New York City, New York. Officers were hired based upon the successful completion of a pre-employment medical examination and training period and then assigned to a particular facility dependent upon the needs of the TBTA. Data available from personnel records of the TBTA contained name, Social Security Number, sex, date of birth, date of hire, date of separation, and specific work history information identifying the bridge(s) or tunnel(s) at which the officers had worked. We obtained information on race from the Social Security Administration. Bridge and

tunnel officers who left employment prior to 1952 were excluded from the cohort since their Social Security Numbers generally were not available from employment records making vital status follow-up very difficult. The primary duties of the bridge and tunnel officers included toll collections from booths, traffic observation within and outside the tunnels, and direction of traffic within the tunnels and on the bridges when necessary (i.e. during rush hours or motor vehicle accidents).

Carbon Monoxide Exposure

Continuous monitoring of CO within the tunnels operated by the TBTA began in 1940 at the Queens Midtown Tunnel and in 1950 at the Brooklyn-Battery Tunnel, and was first summarized in 1958 showing peak measurements exceeding 400 parts per million (ppm). In 1961, at the request of the TBTA, an investigation of the ventilation systems demonstrated 24-hour average CO concentrations inside the tunnels of 53 ppm in the summer (with peaks of 200-300 ppm) and 49 ppm in the winter (with peaks of 100-200 ppm).[26] In 1968, 24-hour average CO concentrations measured inside the tunnels were 35-40 ppm[27]; ranges of CO exposures taken during rush hour traffic were found to range from 120-165 ppm in the morning and 65-145 ppm in the evening in the tunnel toll booths, and 15-45 ppm in the morning and 12-22 ppm in the evening in the bridge toll booths.[28] In 1970, monitoring over 38 continuous days revealed mean CO levels of 63 ppm in the tunnel toll booths and 13 ppm in the bridge toll booths.[29] During the same year, fresh-air ventilation systems were installed in all toll booths. In 1971 an increase in electrical service to

the ventilation fans in the tunnels yielded an increase of approximately 15 percent in tunnel ventilation capacity. Also, in 1971, officers were allowed one 1/2-hour "air break" for each day's work which consisted of two, two-hour tours inside the tunnel. In 1977, ventilation equipment for the tunnels was linked electrically to continuously reading CO monitors. In 1981, daily sampling conducted over two weeks by NIOSH industrial hygienists using direct-reading portable Ecolyzers for area exposures and Draeger direct-reading long duration detector tubes for personal and area exposures found mean area levels of CO of 38.3 ppm inside the tunnels and 23.0 ppm outside the bridge toll booths, and personal samples of 10.8 ppm collected on tunnel officers and 6.2 ppm collected on bridge officers.[30] Peak CO levels measured in the traffic lanes of both the tunnel and the bridge and on the tunnel catwalk were frequently greater than 100 ppm and occasionally greater than 400 ppm. Exposures to nitrogen dioxide, polycyclic aromatic hydrocarbons, particulates, various metals (including lead), and asbestos were also measured and found to be well below the respective permissible or recommended exposure levels.

Smoking histories were collected during medical evaluations of active officers from 1972 to 1981[31] and by NIOSH investigators in 1981.[30] Pipe and cigar smokers were classified as nonsmokers since they experience only a small increase in mortality from coronary heart disease above that of nonsmokers.[21] Carboxyhemoglobin levels measured in 1970 (before ventilation systems were installed in the toll booths) averaged 2.12 and 3.90 percent in nonsmokers and smokers, respectively, for bridge officers; and 2.93 and 5.01 percent in nonsmokers and smokers, respectively, for tunnel officers.[31]

COHb levels measured by NIOSH in 1981 were not found to be significantly different between bridge and tunnel officers, with pre- and post-shift COHb levels rising about 20 percent in nonsmokers and 10 percent in smokers.[30]

Ascertainment of Vital Status

Vital status of each officer was ascertained as of December 31, 1982 through the sources of the Social Security Administration, Internal Revenue Service and National Death Index, among others. For deceased officers, death certificates were obtained from the appropriate state vital statistics offices. The underlying and contributory causes of death were coded by a qualified nosologist according to the rules of the revision of the International Classification of Diseases (ICD) in effect at the time of death.

Statistical Analyses

The bridge and tunnel officer cohort was analyzed using a modified life table analysis system,[32] which computed expected number of deaths using indirect adjustment by multiplying the person-years at risk (PYAR) of dying among the officers by the corresponding age, sex, calendar-time, and cause-specific mortality rates of the New York City population. Accumulation of PYAR and observed deaths began with the first day of employment at the TBTA on or after January 1, 1952 and continued until either December 31, 1982 or the date of death of the officer, whichever occurred earlier. The relative risk estimates

for cause-specific mortality among the cohort were calculated as standardized mortality ratios (SMRs) which were derived by dividing the observed deaths by the number expected. One-sided significance tests and 90 percent confidence intervals were computed for the SMRs assuming a Poisson distribution for the observed deaths.[33] SMRs were considered to be statistically significant if the P values were less than 0.05 or, equivalently, if the lower confidence limit was greater than 1.00.[34]

We examined the mortality experience of those officers employed only in tunnels and those officers employed only on bridges, separately. This was done since previous environmental sampling had indicated that CO levels had been substantially higher within and around the tunnels than on bridges (see Methods-carbon monoxide exposure). Because environmental sampling results for CO were only available for a few years of the study, duration of employment was used as a surrogate for cumulative exposures. Two categories of less than, or equal to and greater than 10 years employment were used in an attempt to ascertain the adverse effects from cumulative long-term CO exposures. For cancers only, an additional analysis by latency (time period from initial employment to death) was performed.

In order to make direct comparisons of the mortality experience of bridge officers as compared with tunnel officers while controlling potential confounders and effect modifiers, the Cox proportional hazards model with time-dependent covariables was also employed to analyze the mortality experience of the cohort. This analysis uses internal comparisons and,

therefore, did not require the use of mortality rates from New York City. A recent modification to the BMDP2L survival analysis procedure permitted general forms of the Cox model to be used.[35] This method produces relative risk estimates based upon incidence rates as a function of covariables such as age, exposure status, calendar time, and duration of employment which may change over the period of study.

RESULTS

Population Identification

There were 4,317 bridge officers and 1,212 tunnel officers employed between January 1, 1952 and February 10, 1981 at the Triborough Bridge and Tunnel Authority (TBTA) as shown in Table 1. The cohort contributed a total of 103,900 person-years at risk. As of December 31, 1982, 88% of the cohort were alive, 9% were deceased, and 3% were lost to follow-up. Death certificates were obtained for 97% (460 out of 474) of all known deaths. Almost twice the percentage of tunnel officers died (13%) as compared with bridge officers (7%). On average, as shown in Table 2, the bridge officers and tunnel officers were very similar in racial composition and calendar year when born. In addition, the bridge officers and tunnel officers started employment at the TBTA, and died at approximately the same age and calendar-time period. However, on average, the tunnel officers had worked two years longer at the TBTA than did the bridge officers.

Standardized Mortality Ratio Analyses

As Table 3 shows, in the 31-year period between January 1, 1952 and December 31, 1982, the overall mortality among bridge officers was less than expected -- 314 deaths observed, as compared with 409 expected, standardized mortality ratio (SMR) = 0.76 -- when compared to the mortality experience of the New York City population. The overall mortality among tunnel officers was approximately equal to that expected -- 160 deaths observed as compared to 153 expected, SMR = 1.04. Heart disease mortality among tunnel officers was the only cause of death that was statistically significantly elevated among the entire bridge and tunnel officer cohort.

Table 3 shows there were 67 deaths among tunnel officers from heart disease [International Classification of Diseases (ICD) 400 to 468 in the sixth and seventh revisions and ICD 390 to 458 in the eighth and ninth revisions excluding 430-438] as compared to 54 expected, an excess of 24 percent. This excess was even more pronounced among tunnel officers for deaths due to arteriosclerotic heart disease (ASHD) (ICD 420 in the sixth and seventh revisions and ICD 410 to 414 in the eighth and ninth revisions), 61 deaths observed compared to 45 expected, a 35 percent increase . The mortality from ASHD increased to 88 percent over expected among tunnel officers employed for more than 10 years at the TBTA, 30 deaths observed as compared to 16 expected.

Table 4 shows the risk of ASHD mortality by age at death among tunnel officers and bridge officers. Prior to age 40 among tunnel officers, there were no deaths attributable to ASHD. After age 60, the relative risk of ASHD was found to be 1.0. However, during the working years, among tunnel officers, the relative risk of ASHD increased from 32 percent over expected for the age group 40-49, to 74 percent over expected for the age group 50-59. No elevation in risk of mortality by age was observed among bridge officers with the exception of those who had died prior to age 40. All tunnel officers who died from ASHD had started employment at the TBTA prior to 1960 (mean year = 1951) and all were employed during the decades of the 50's and 60's when exposures to CO were considerably higher than in the 70's (See METHODS - carbon monoxide exposure).

To help evaluate whether the excess risk from heart disease could have been related to smoking, we examined the other main smoking-related cause of death, lung cancer. Mortality from cancer of the lung was lower than expected among both bridge officers (18 deaths observed compared to 21 expected; SMR = 0.85) and tunnel officers (9 deaths observed compared to 9 expected; SMR = 0.97) as shown in Table 3. The power of this study to detect a two-fold increase in lung cancer for the cohort was at least 99% and among tunnel officers was 80% assuming an alpha level of 0.05 and a one sided test. No substantial elevation in mortality from lung cancer was evident for either subgroup with increasing duration of employment or with increasing time period since first employment. Also shown in Table 3 is a deficit of deaths from "all other causes" among both bridge officers and tunnel officers.

Cox Proportional Hazards Model

Due to the significant excess risk from ASHD among tunnel officers observed in the SMR analysis, the Cox proportional hazards model was used to examine this finding in greater detail. Potential confounding and effect modification were examined by including duration of employment, age, and calendar time and their interactions in the models.

Similar to the effect suggested by the SMR analysis, a direct comparison of job location (bridge versus tunnel) showed significant elevation in risk from ASHD for tunnel officers when compared to bridge officers, adjusted for duration of employment, age and calendar time (RR = 1.54, P = 0.01). In order to investigate the potential effect of chronic exposure to carbon monoxide, trends in ASHD mortality with number of years employed were modeled. In contrast to the results of the SMR analysis, no significant trend was found with duration of employment in any of the models used. However, when the interaction of job location with age was examined, the result was found to be statistically significant (P = 0.015). Specifically, the risk of ASHD in tunnel officers increased approximately 5.5% per year of age relative to bridge officers, starting at age 45 when the risk for the two groups was essentially the same.

Cognizant of the important finding of Rosenberg et al.[36] and others[15-22,37] that the substantial excess risk from heart disease decreases rather quickly after cessation of cigarette smoking, we chose to test a similar hypothesis in this study. Tunnel officers were directly compared to bridge officers with

regard to their ASHD mortality experience after cessation of exposure, defined as that period of time following date last employed. The time following cessation of employment was stratified into four intervals: 0 to 1 month, 2 to 23 months, 2 to 4 years, and 5 or more years. The category 0 to 1 month was chosen in order to distinguish the officers who either died on the job or shortly thereafter, from individuals who died after cessation of employment and hence after exposure to the workplace environment. A direct comparison of tunnel to bridge officers in each time interval at each of three ages is presented in Table 5. This table demonstrates that the risk of ASHD among tunnel officers relative to bridge officers increases with age. However, after separation from employment for as little as five years, the elevated risk of ASHD mortality among tunnel officers declines and begins to approach that of bridge officers for each of the ages. These comparisons were intended to remove any unknown risk factors associated with time since separation from employment including the possibility that cardiovascular morbidity may be a precipitant of termination. All results were adjusted for calendar time and duration of employment.

In order to investigate the effect of lowering of exposure which began after 1970 with the addition of fresh air ventilation in all tunnel booths along with an increase in tunnel ventilation, trends in ASHD mortality after that time were modeled. A significant decrease in relative risk of ASHD mortality was found ($P = 0.042$). Specifically, the risk of ASHD in tunnel officers was modeled as an exponential decline of 6.4% per year after 1970 when compared to the lesser-exposed bridge officers. Figure 1 illustrates this decline in relative risk after 1970 for tunnel officers at 45, 55 and 65 years of age.

DISCUSSION

The present results suggest that exposure to carbon monoxide (CO) may be an important factor in arteriosclerotic heart disease (ASHD) mortality. In our study, tunnel officers employed by the Triborough Bridge and Tunnel Authority (TBTA) experienced a 35% excess risk of ASHD mortality as compared to the New York City population. The data suggest that two factors interacted to produce the elevated risk of heart disease: the higher levels of exposure to CO experienced by the tunnel officers, as well as the movement into a critical higher age group. Both apparently act together to produce a significantly elevated risk of ASHD mortality.

In attempting to understand the etiology of ASHD in this cohort, another finding is of particular interest. The excess risk of ASHD mortality in tunnel officers when compared to the lesser exposed bridge officers dropped in all age groups after cessation of exposure when individuals left employment in the tunnels. In addition, a similar reduction in risk was found after 1970 when substantially more ventilation was introduced, thereby reducing exposures to CO. This effect suggests that regardless of age, the adverse health effects experienced by the tunnel officers at the TBTA are, in large part, reversible. Similar results to these have been observed among cigarette smokers in whom the excess risk from heart disease morbidity and mortality begin to decrease after cessation of smoking with much of the risk dissipating within 2-10 years.[15-22,36,37]

Carbon monoxide may contribute to heart disease mortality through either the effects of current exposure, or as the result of the accumulation of exposure over a sustained period. Length of exposure was not a risk factor for heart disease mortality in this study suggesting that the acute effects of exposure to carbon monoxide are more important than the chronic. These results are consistent with studies of the effect of cigarette smoking on heart disease mortality which also demonstrate that it is the number of cigarettes smoked, not the length of years of cigarette smoking that is associated with the excess risk of heart disease mortality.[38,39].

Prior to 1970, it had been documented that CO levels in the tunnels averaged over 50 parts per million (ppm), the current Occupational Safety and Health Administration (OSHA) standard, with excursions frequently exceeding 400 ppm. These excursions were due to the congestion of cars within the tunnels caused by motor vehicle accidents and rush-hour traffic and by the limitations of the ventilation systems. This level of exposure to CO for tunnel officers would result in a range of carboxyhemoglobin (COHb) that is close to the COHb level observed in cigarette smokers[2,23-25] who are also exposed to an average CO level estimated at 50 ppm[40] with excursions up to 475 ppm[41] and who are also known to experience a greater risk of heart disease.[15-22]

Mortality from ASHD has a complex multifactorial etiology. The presence of other risk factors in addition to occupational CO exposures, including cigarette smoking, hypertension, hyperlipidemia, family history of heart disease, marked obesity, socio-economic status, and sedentary living increases the risk of developing ASHD.[42-44] Since this study was conducted using employment records, information concerning many of these risk factors for the bridge and tunnel officers was limited. However, inferences about some of these risk factors can be made from the results of this study and other information.

Cigarette smoking, uncontrolled in the analysis, could explain some of the excess risk for ASHD if the officers' smoking habits were substantially different from that of the comparison population.[45] Although the tobacco smoking habits of the officers had not been recorded at the time of employment, information on smoking status of many active members of this cohort had been collected in two separate studies.[30,31] Among bridge officers, the percentages of current smokers, exsmokers, and nonsmokers were 43.1, 28.6 and 28.3, respectively. Among tunnel officers, the percentages were almost identical, 43.7, 28.6 and 27.7, respectively. These percentages resemble the proportion of smoking habits among white males in the general population during the 1970's[46] of 41.4%, 29.9%, and 28.7%, respectively.[47] Apparently, if this cross-sectional view of the smoking habits obtained is reasonably representative of the cohort as a whole, then the smoking habits among the bridge officers and tunnel officers were not substantially different from each other or from the general population. Thus,

smoking probably cannot explain the substantially increased risk of ASHD mortality seen among tunnel officers of the TBTA. Furthermore, if smoking among this cohort had been higher than that of the general population, a significant excess of lung cancer mortality among bridge officers and tunnel workers would also have been expected since smoking is nearly seven times more strongly associated with lung cancer than with ASHD.[16,21] No increase in lung cancer mortality, however, was observed among either sub-cohort. Also, no increase in deaths from emphysema was found.

There were no deaths attributable to hypertensive heart disease among tunnel officers (compared to 1.6 deaths expected), and the mortality risk from stroke was lower than expected based on the New York City population (SMR = 0.92). Since hypertension and cigarette smoking are the major risk factors for stroke,[48] the absence of elevated mortality from this disease suggests that neither hypertension nor cigarette smoking substantially contributed to the excess mortality due to ASHD.

Selection bias is also unlikely to explain these results. Self selection into jobs by individuals with increased risk from ASHD was observed in studies of London bus drivers and collectors.[49] However, the TBTA employment practices were such that a recruit would not know his job location until after finishing the pre-employment physical examination and training. There was no difference in the qualifications for bridge officers and tunnel officers. Thus, the requirements of the TBTA rather than the officers' preferences determined their worksite selection. The TBTA requirements would be determined by the

turnover of employees which was similar in both bridge and tunnel officers. However, to limit the possibility of selective migration between bridges and tunnels for officers with an increased risk of ASHD, we eliminated from the study those officers whose personnel records indicated transfer between job locations. In addition, the duties of the bridge officers, mainly toll collection, are more sedentary than the duties of the tunnel officers who must also man the observation booths within the tunnels. Thus, if preference of the officer was a major factor in initial or future worksite selection those less fit, due to obesity or other factors, would be most likely attracted to the bridges rather than tunnels.

Finally, bridge officers and tunnel officers are drawn from the same applicant pool, and hence the same socio-economic class which makes it less likely that there would be such great differences between the two groups with regard to factors such as race, dietary habits, alcohol consumption, and physical activity during leisure time.

In the absence of data to implicate other causes for the increased mortality risk from ASHD among tunnel officers, the available evidence suggests that the factor was directly associated with employment. The most likely factor would be exposure to carbon monoxide. However, the possibility should be considered that one of the other constituents of vehicular exhaust (e.g., nitric oxides, hydrocarbons, particulates, lead, sulfur, aldehydes) or some other characteristic of the worker environment (e.g., noise or stress) could

have played a role in aggravating cardiovascular disease, although the implication of these other factors and their effect upon the heart is highly speculative.[50-52]

This study had some potential strengths and limitations that should be considered. First, the choice of the comparison population used may have underestimated the true risk of mortality in this study. The New York City population, while more appropriate than the U.S. as a basis for calculating expected deaths, like most generalized populations, includes numbers of chronically ill or otherwise unemployable persons. Since most populations of workers are healthier than that of the general population, comparison of the mortality experience of bridge officers and tunnel officers to that of a general population may produce an apparent deficit in mortality, as seen among bridge officers, the so-called "healthy worker effect".[53] However, the use of the bridge officers in the proportional hazards model was considered to be an ideal internal comparison group. Bridge officers are drawn from the same applicant pool as tunnel officers, undergo the same pre-employment selection process, have comparable duties, and belong to the same socio-economic class. Second, in lieu of detailed monitoring data of past environmental CO exposures for many years of the study, we had to define exposure by classifying officers according to the duration of their employment and their assignment to either bridge or tunnel work which are crude surrogate measures of the actual exposure. Actual measurements throughout the years of the study would have been preferable to define more precisely the excess risk from ASHD mortality. Third, ascertainment of vital status among our cohort was 97 percent complete

and, while this percentage of follow-up is well within the range generally considered acceptable for cohort mortality studies, the 3 percent deficit had the effect of inflating person-years at risk and thus lowering the SMRs. Furthermore, death certificates were located and cause of death recorded for 460 (97 percent) of the 474 known deaths. This had the effect of lowering the cause-specific SMRs by an additional three percent, on the average. These last two factors could have contributed to an underestimation of the magnitude of the ASHD mortality risk from employment at the TBTA. Finally, since this is a study of mortality as ascertained from death certificates we are unable to assess the accuracy of the stated diagnoses or define the date of myocardial infarction in contrast to the date of death.

In summary, this study indicates that occupational exposures to carbon monoxide contributes to ASHD mortality. The excess risk in cardiovascular disease mortality that we observed may have been caused by long-term continuous lower exposure to carbon monoxide, short-term repeated peak exposures or a combination of both. However, the absence of any significant relationship between duration of employment and ASHD mortality tends to discredit the contribution of long-term exposures. The significant effect of age on ASHD mortality implies that age is an essential contributing factor in the excess risk of ASHD. The adverse effects of carbon monoxide poisoning on the risk of ASHD mortality seem to be reversible upon cessation of exposure.

We believe our results have three important implications. First, given the magnitude of the effect that we have observed for a very prevalent cause of

death, exposure to CO, in combination with underlying heart disease or other cardiovascular risk factors could be responsible for a very large number of preventable deaths. Second, in parallel to the effect of cessation of cigarette smoking on the risk of cardiovascular disease, cessation of occupational exposure to carbon monoxide results in a decline in risk for heart disease mortality. Third, the parallel findings of this study of occupational exposure to CO and those studies of the relationship of cigarette smoking to cardiovascular mortality suggest that CO may play an important role in the pathophysiology of cardiovascular mortality associated with cigarette smoking.

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Table 1. Vital status among male Bridge Officers and male Tunnel Officers at the Triborough Bridge and Tunnel Authority, New York City, New York as of 12/31/82

VITAL STATUS	Bridge officers		Tunnel officers		Total	
	No of officers (%)					
Alive	3872	(90)	1014	(84)	4886	(88)
Deceased	314	(7)	160	(13)	474	(9)
Death certificates obtained	303		157		460	
Death certificates outstanding	11		3		14	
Lost to follow-up	131	(3)	38	(3)	169	(3)
Total	4,317	(100)	1,212	(100)	5,529	(100)
Person-years at risk	79,865		24,035		103,900	

Table 2.

Demographic characteristics among male Bridge Officers and male Tunnel Officers

Triborough Bridge and Tunnel Authority, 1/1/52 - 2/10/81, New York City, New York

Demographic Characteristics*	Bridge officers	Tunnel officers
Number	4,317	1,212
% White	83.1	80.1
Year of Birth	1936	1932
Year First Employed	1963	1961
Year Last Employed	1966	1966
Age First Employed	26 yrs.	29 yrs.
Duration of Employment	35 mo.	61 mo.
Number Deceased (%)	314 (7%)	160 (13%)
Age Deceased	49	51
Year Deceased	1974	1974

*Mean Values

Table 3. Mortality (1952-1982) according to duration of employment among male Bridge Officers and male Tunnel Officers, Triborough Bridge and Tunnel Authority, New York City, New York[a]

BRIDGE OFFICERS

CAUSES OF DEATH	DURATION OF EMPLOYMENT											
	<10 years				≥10 years				TOTAL			
	Obs	Exp	SMR	90%CI	Obs	Exp	SMR	90%CI	Obs	Exp	SMR	90%CI
All Heart Disease	78	96	0.82	0.67,0.99	30	33	0.91	0.65,1.23	108	129	0.84	0.71,0.99
ASHD[b]	66	76	0.87	0.70,1.07	23	28	0.81	0.56,1.15	89	104	0.85	0.71,1.02
Lung Cancer	13	16	0.83	0.49,1.32	5	6	0.91	0.36,1.91	18	21	0.85	0.55,1.26
All Other Causes	154	223	0.69	0.60,1.79	34	37	0.93	0.69,1.24	188	259	0.73	0.64,0.82
All Causes	245	334	0.73	0.66,0.82	69	75	0.92	0.75,1.12	314	409	0.76	0.70,0.84

TUNNEL OFFICERS

CAUSES OF DEATH	DURATION OF EMPLOYMENT											
	<10 years				≥10 years				TOTAL			
	Obs	Exp	SMR	90%CI	Obs	Exp	SMR	90%CI	Obs	Exp	SMR	90%CI
All Heart Disease	35	36	0.98	0.72,1.30	32	19	1.72++	1.25,2.31	67	54	1.24+	1.01,1.51
ASHD[b]	31	29	1.07	0.77,1.44	30	16	1.88++	1.36,2.56	61	45	1.35+	1.09,1.68
Lung Cancer	5	6	0.83	0.33,1.75	4	3	1.29	0.44,2.95	9	9	0.97	0.52,1.73
All Other Causes	69	68	1.01	0.82,1.23	15	21	0.72	0.44,1.11	84	89	0.94	0.78,1.13
All Causes	109	110	0.99	0.84,1.16	51	43	1.20	0.94,1.52	160	153	1.04	0.92,1.20

a OBS = observed number of deaths; EXP = expected number of deaths; SMR = standardized mortality ratio (Obs/Exp); CI = confidence interval. Expected number of deaths are based on the death rates for New York City rounded to the nearest whole number.

b ASHD = arteriosclerotic heart disease

\+ Significantly different from 1.00 ($P < 0.05$)

++ Significantly different from 1.00 ($P < 0.01$)

Table 4. Mortality (1952-1982) from Arteriosclerotic heart disease (ASHD) according to age among male Bridge Officers and male Tunnel Officers, Triborough Bridge and Tunnel Authority, New York City, New York[a]

	Bridge Officers				Tunnel Officers			
Age	OBS	EXP	SMR	90% CI	OBS	EXP	SMR	90% CI
<40	12	6	1.79+	1.15,3.24	0	2	-	-
40-49	28	29	0.97	0.69,1.32	15	11	1.32	0.84,2.10
50-59	36	38	0.96	0.70,1.25	34	20	1.74+	1.25,2.26
60+	13	31	0.42	0.25,0.67	12	12	1.00	0.58,1.62
All Ages	89	104	0.85	0.71,1.02	61	45	1.35+	1.09,1.68

a OBS = Observed number of deaths; EXP = expected number of deaths; SMR = standardized mortality ratio (OBS/EXP); CI = confidence interval. Expected number of deaths are based on the death rates for New York City rounded to the nearest whole number.

\+ Significantly different from 1.00 ($P < 0.05$)

Table 5. Risk Estimates[a] of ASHD Mortality by Time Since Last Employment and Age among male Tunnel Officers relative to male Bridge Officers

Triborough Bridge and Tunnel Authority, New York City, New York

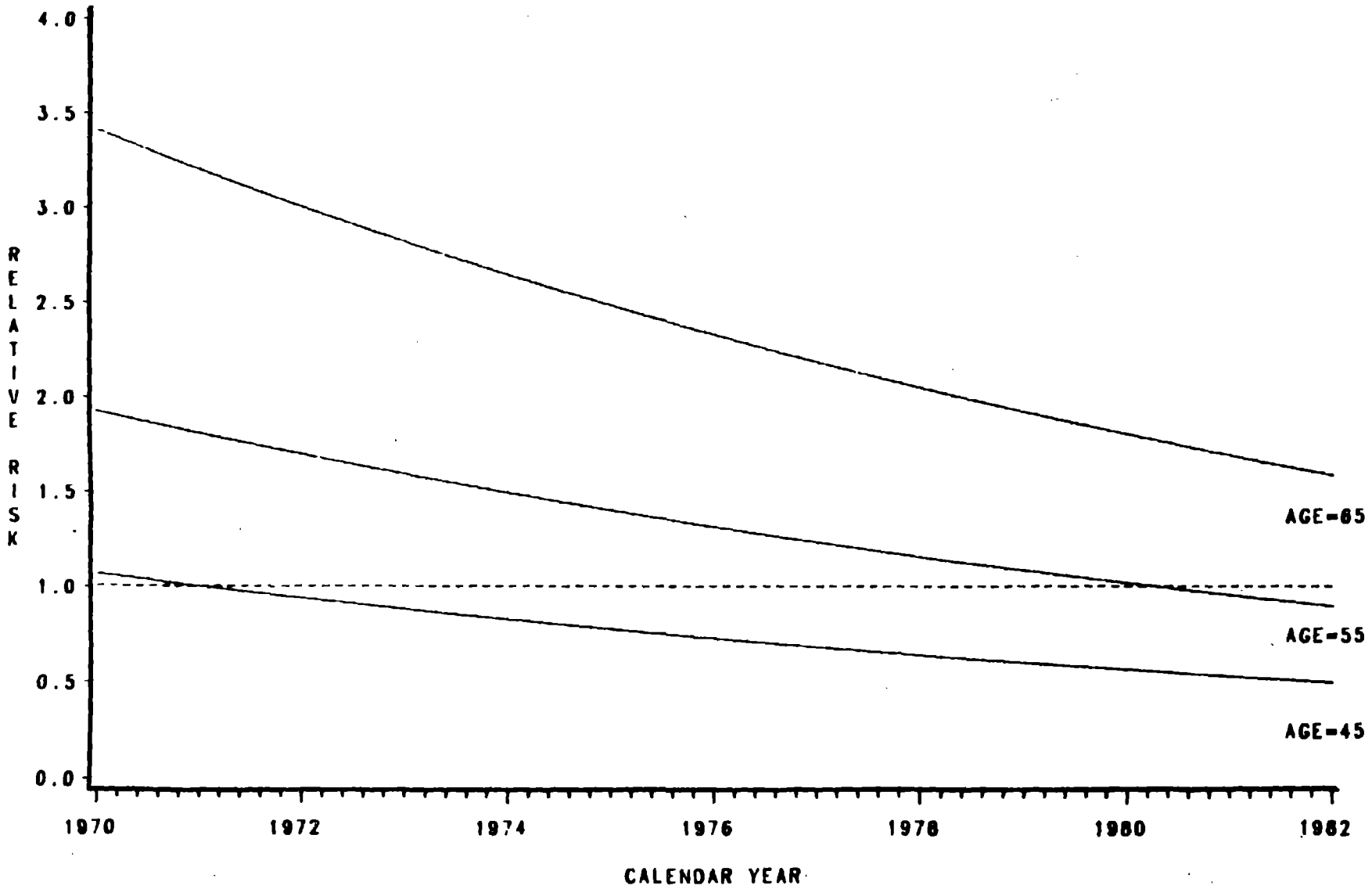
	Time Since Last Employment			
	0-1 mo.	1-23 mo.	2-4 yr.	≥ 5 yr.
Age	RR[a] (90% CI)	RR (90% CI)	RR (90% CI)	RR (90% CI)
45	0.98 (0.51-1.89)	1.39 (0.53-3.63)	2.11 (0.68-6.58)	0.94 (0.59-1.50)
55	1.59 (0.85-2.95)	2.25 (0.88-5.76)	3.41+ (1.11-10.43)	1.51 (0.99-2.30)
65	2.57+ (1.18-5.58)	3.63+ (1.27-10.34)	5.53++ (1.65-18.56)	2.45+ (1.31-4.58)

a RR, Relative Risk Estimates using Cox proportional hazards model

\+ Significantly different from 1.00 ($p < 0.05$)

++ Significantly different from 1.00 ($p < 0.01$)

Figure 1. Risk of ASHD Mortality in Tunnel Officers Relative to Bridge Officers as a Function of Calendar Year after the Introduction of Ventilation in 1970.



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15. Supplementary Notes

16. Abstract (Limit: 200 words) A retrospective study was conducted of 5529 New York City bridge and tunnel officers to determine the effect of occupational exposure to carbon-monoxide (630080) (CO) with specific reference to heart disease mortality. Continuous monitoring of CO within the tunnels operated by the Triborough Bridge and Tunnel Authority began in 1940. In 1961 the 24 hour average CO concentrations inside the tunnels were 53 parts per million (ppm) in the summer and 49ppm in the winter with peaks of 200 to 300ppm in the summer and 100 to 200ppm in the winter. Monitoring in 1970 indicated CO levels of 63ppm in tunnel toll booths and 13ppm in bridge toll booths. In 1977 ventilation equipment which had been installed was linked to continuously reading CO monitors. Peak 1981 CO exposures in the traffic lanes of both the tunnel and bridge and on the tunnel catwalk were often greater than 100ppm and sometimes surpassed 400ppm. Arteriosclerotic heart disease (ASHD) was the cause of death in 61 former tunnel officers as compared to the expected number of 45. A Cox proportional hazards model was used to compare the risk of mortality from ASHD among tunnel officers to the less exposed bridge officers. While there was a significant association between exposure and age, no association of ASHD was noted with length of exposure. After cessation of exposure the elevated risk of ASHD among tunnel officers as compared to bridge officers declined, usually within as short a time period as 5 years. According to the authors, present research shows that CO may be important in the pathophysiology of cardiovascular mortality associated with cigarette smoking.

17. Document Analysis a. Descriptors

b. Identifiers/Open-Ended Terms NIOSH-Publication, NIOSH-Author, Cardiovascular-system-disorders, Air-quality-monitoring, Air-sampling, Tunnel-workers, Automotive-exhausts, Automotive-emissions, Toxic-gases, Epidemiology

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